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## **Approach to syncope in the Emergency Department**

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## **Approach to syncope in the Emergency Department**

### **Abstract (91 words)**

Syncope is a common reason for Emergency Department (ED) attendance and it presents a major management challenge with regard to the appropriate work-up and disposition. Nearly 50% of patients are admitted, and for many this is unnecessary; Clinical decision rules have not proven to decrease unnecessary admissions. The European Society of Cardiology has recently developed guidance for managing syncope in the ED. This article highlights the key steps in evaluating syncope in the ED, factors involved in determining risk of a cardiac cause, and considerations for admission, observation or discharge.

## **Approach to syncope in the Emergency Department**

Syncope is a common reason for ED attendance and it presents a major management challenge with regard to the appropriate work-up and disposition. There is a lack of high-quality evidence-based strategies to enable clinicians to determine which patients have benign causes, are at high risk of short-term adverse events or at high risk of long-term adverse outcome. Despite a relatively low incidence of short-term adverse events (**Table 1**), admission rates remain high with limited alternative strategies. This is due, in most hospitals, to a lack of a clear lead specialty, specialist syncope experts, specialist ambulatory syncope units and specialist outpatient syncope clinics [1]. Whilst those who attend the ED are likely to represent the more extreme end of the syncope spectrum some patients with high-risk features may attend General Practice (GP). This article may also be useful to help guide GP referrals to routine or urgent rapid access syncope clinics or cardiology outpatient services. The majority of patients who either visit their GP or who do not seek any medical attention are more likely to be younger and more likely to have had an episode of reflex syncope [2].

### **Case examples**

#### *Case 1*

A 75-year-old male presents to the ED having experienced a sudden Transient Loss Of Consciousness (TLOC) whilst waiting for a bus with his 8-year-old granddaughter. He fell to the floor and a passer-by phoned for an ambulance, as he was slow to recover even after 10 minutes. He has treated moderate hypertension but otherwise no other previous medical history. He recalls feeling lightheaded, sweaty and

nauseated for several minutes prior to the collapse and his 8 year old granddaughter recalls him looking pale and not responding to her for a minute or so prior to the collapse and then having a short lived episode of shaking immediately after the collapse. In the ED he feels back to normal but still feels a little confused about the incident. Physical examination is normal. The ECG recorded on arrival shows 1<sup>st</sup> degree heart block.

### *Case 2*

A 75-year-old male presents to the ED having experienced a sudden TLOC whilst driving. He collided at slow speed with a lamppost and a passer-by phoned for an ambulance. He has treated mild hypertension but otherwise no other previous medical history. He has no recollection of the incident or the moments preceding it. In the ED he feels back to normal. Physical examination is normal. The ECG recorded on arrival shows 1<sup>st</sup> degree heart block.

### *Case 3*

A 45-year-old male presents to the ED having experienced a sudden TLOC whilst carrying a cup of tea across his kitchen. He fell to the floor and was found by his wife who heard a crash from the room next door. He recovered within 5 minutes and was brought to the ED by his wife. He has had one previous episode of transient loss of consciousness 3 weeks prior. He has no previous medical history. Physical examination reveals superficial burns to his anterior chest wall, but cardiovascular exam is normal. The ECG recorded on arrival shows sinus rhythm without evidence of ischemia or conduction disturbance.

## **Is this syncope?**

All 3 patients have undoubtedly had an episode of Transient Loss of Consciousness; TLOC. The two commonest causes for this are syncope and neurological seizure. Differentiation of the two is not always straightforward; the 2018 European Society of Cardiology (ESC) Guidelines for the diagnosis and management of syncope highlight the difficulty of diagnosing TLOC as being of syncopal origin (i.e. due to cerebral hypoperfusion) in the ED [2]. SYNERGI (SYNcope Expert Research Group International) [3] suggest a pragmatic definition of syncope: 'a transient loss of consciousness, associated with inability to maintain postural tone and with immediate spontaneous and complete recovery'. [4] A very careful history is needed to differentiate syncope from epilepsy and other non-TLOC conditions such as pre-syncope, light-headedness, vertigo, disequilibrium, mechanical and collapse (i.e. loss of postural tone). In the absence of witnesses, information from the patient regarding prodrome, provocation and prior history can be useful; information from witnesses, particularly on the time to recovery will be extremely helpful. Where paramedics are involved, examine the ambulance notes for initial observations and review any pre hospital ECG. These are a great source of useful information that can be hard to locate later down the line.

In most cases the ED clinician can establish the presenting complaint of syncope. ED clinicians should not label TLOC patients as 'collapse query cause'. This implies a lack of attention to the history of the event and leads to poor patient management, treatment and disposition decisions.

Pre-syncope is the feeling of being about to pass out without actual Loss of Consciousness (LOC). Pre-syncope has ordinarily thought to be associated with a better prognosis compared with syncope and should be classified separately. However some recent studies have suggested that patients presenting with pre-syncope may have outcomes similar to those observed in patients with syncope [5-7] and the recent ESC guidelines [2] suggest that in the ED, presyncope should be managed similarly to syncope as it carries the same prognosis.

Case 1 presents the most challenging distinction between syncope and neurological seizure. However syncope is more likely. It is common for syncope patients to have short-lived seizure like activity (anoxic seizure) [8]. The presence of a prodrome of light-headedness, feeling of warmth and sweating makes syncope much more likely. [9,10]

### **Is there a serious underlying diagnosis?**

In the ED, once the presenting complaint of syncope is established, a serious underlying diagnosis must next be sought. It is essential to identify conditions such as ruptured abdominal aortic aneurysm and severe upper gastrointestinal bleeding that if undetected, can cause rapid deterioration. An underlying diagnosis can be identified in the ED in around 50% of patients. Of the underlying diagnoses that are serious, non-cardiovascular (i.e. pulmonary embolus/ruptured abdominal aortic aneurysm/upper gastrointestinal bleeding/subarachnoid haemorrhage) are more likely to be recognised in the ED than cardiovascular conditions especially underlying arrhythmia (unless present on admission ECG). [11] If a precipitating diagnosis is



found, management of the patient should follow the recommended practice for that condition.

### **What is the risk of a serious outcome in patients with syncope?**

If an underlying diagnosis cannot be identified in the ED, subsequent management will be guided by assessment of the risk of a serious outcome, notably a future major cardiovascular event or sudden cardiac death. Risk stratification includes determining the type of syncope and the patient's risk factors for a cardiac event.

There are three main categories of syncope. A patient thought likely to have a reflex or postural categorisations will be at low-risk of serious outcome. A patient thought likely to have a cardiac categorisation will be at high-risk of serious outcome. **Table 2** details the main categories of causes of syncope grouped by common pathophysiology, presentation and risk.

The 2018 ESC Guidelines for the diagnosis and management of syncope [2] provides a list of high and low risk features that can be used for ED risk stratification [Table 3]. Once ED risk stratification has been undertaken the ESC ED risk stratification flowchart [Figure 1] should be used to determine subsequent management [2].

### **Patients with low-risk features**

Patients with **Table 3** low-risk features only are likely to have reflex or orthostatic syncope. The syncopal event will include an associated prodrome or typical precipitating event (e.g. a sudden unexpected unpleasant sight or sound, or

prolonged standing), the patient's past medical history may include a long history of recurrent syncope with low-risk features and an absence of structural heart disease. Physical examination and ECG will be normal. Reflex syncope generally confers an excellent prognosis, [12] orthostatic syncope is also low-risk but may carry a slightly poorer prognosis than reflex or situational syncope due to comorbidities. [13]

A patient with only low-risk characteristics and without any high-risk characteristics can be discharged safely from the ED with a likely diagnosis of reflex or orthostatic syncope. They can be managed with adequate patient education that may be started in the ED and may benefit from a low-risk syncope advice sheet [14] and reassurance and/or education that can be provided by their GP.

Some patients with episodes causing injury or frequent episodes may benefit from referral to a specialist syncope clinic and need further investigation to guide specific treatment. Examples here include pacemaker insertion in cardioinhibitory reflex syncope or drug treatment in vasodepressor reflex syncope. In the event of associated injury or social or welfare reasons, some may require admission to hospital. However in general, admission to hospital for patients with low-risk features is inefficient as they can be safely discharged home from the ED, significantly reducing hospital admissions, costs and adverse outcomes associated with unnecessary admission.

### **Patients with high-risk features**

These patients will have no associated prodrome or typical precipitating event, a past medical history including structural heart disease or an abnormal physical

examination or ECG **[Table 3]**. They are at risk of cardiac syncope.

Structural heart disease **[15-20]** and primary electrical disease **[21]** are major risk factors for sudden cardiac death and overall mortality in patients with syncope. They may require urgent advanced investigation such as echocardiography, ECG monitoring, specialised cardiovascular tests and review from an expert in syncope +/- treatment. They must not be discharged from the ED unless this can occur during the ED stay, in a syncope clinical decision/investigation unit or in a rapid follow-up clinic. The optimum duration of ECG monitoring after the index episode is unclear but is likely to lie between 4 and 24 hours. **[22,23]**. ECG monitoring should occur in an area where resuscitation facilities are available.

### **Exercise associated syncope**

Exercise associated syncope is defined as syncope occurring during or immediately after exercise. Although most cases are benign, especially those associated with post exercise collapse which are commonly reflex, patients with exercise associated syncope include groups of patients at high risk of sudden death and conditions such as Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) **[24]**, Brugada syndrome and Hypertrophic cardiomyopathy (HCM) **[25]** should be considered. These can present with syncope during exercise without warning.

Arrhythmogenic Right Ventricular Cardiomyopathy is an inherited cardiac disorder associated with paroxysmal ventricular arrhythmias and sudden cardiac death. ECG characteristics include the epsilon wave (a small positive deflection at

the end of the QRS complex, seen in 30% of patients), T wave inversions in V1-3 (85% of patients), prolonged S-wave upstroke of 55ms in V1-3 (95% of patients), localised QRS widening of 110ms in V1-3 and paroxysmal episodes of ventricular tachycardia with LBBB morphology **[Figure 2]**.

Brugada syndrome is an ECG abnormality with a high incidence of sudden death in a patient with a structurally normal heart. There are 3 types, the most common, type 1 is associated with a coved ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave, a pattern that has been referred to as the Brugada sign **[Figure 3]**.

HCM is an inherited cardiac disorder associated with left ventricular hypertrophy (LVH) occurring in the absence of any inciting stimulus such as hypertension or aortic stenosis. The most commonly observed pattern is asymmetrical thickening of the anterior interventricular septum with the ECG showing left ventricular hypertrophy with associated ST segment / T-wave abnormalities and deep narrow Q waves < 40 ms wide in the lateral leads I, aVL and V5-6 **[Figure 4]**.

Patients with exercise associated syncope or suspected Arrhythmogenic Right Ventricular Cardiomyopathy, Brugada syndrome or HCM can be managed in an ED syncope clinical decision/investigation unit and/or a rapid access syncope clinic but if these are not available they are likely to require hospital admission.

### **Syncope with no prodrome**

Patients with trauma (commonly facial due to unconsciousness meaning they are unable to put their hand out) and those without prodromes and/or without apparent triggers and/or atypical presentation (termed non-classical reflex syncope forms) should be considered for further arrhythmia investigation even if they are of younger age. This is because arrhythmic syncope is associated with no or less than 3 seconds of prodrome. On the other hand this prodrome is up to 3 minutes in reflex syncope. Case 3 has no concerning features in the history, exam or ECG albeit that he suffered significant trauma (burns) from the episode. Similarly, in case 2 the patient had no clear trigger, and no prodrome, making this a high-risk event.

### **Patients without high or low-risk features**

These patients will have no low-risk characteristics and none or only minor high-risk characteristics. It will not be clear whether the underlying diagnosis is cardiac, reflex or orthostatic syncope. Case 3 is a good example of such a patient; the only high-risk feature is the lack of prodrome. This patient will require urgent expert syncope opinion probably via a specialist outpatient clinic. [26] They probably don't need to be admitted to hospital unless an ED syncope clinical decision unit or rapid access syncope clinic is not available.

### **Patients with both high and low-risk features**

These patients should generally be managed as high-risk. However if a patient who is high-risk according to past medical history or abnormal ECG presents with a clear benign low-risk story (i.e. the syncopal event is a low-risk three minute prodromal period in which they were pre-syncopal, nauseated and diaphoretic) then they do not

require admission. They will require investigation for any potential underlying condition (e.g. physical examination revealed a likely murmur of aortic stenosis or ECG suggested long QT syndrome) but this is not likely to be the cause of the index event.

### **Does the patient need to be admitted to hospital?**

Many admissions are unnecessary; two thirds of serious outcomes occur whilst the patient is in the ED and the rate of post-ED serious outcome is actually quite low at 3.6% in the following month **[Table 1]**. Currently, approximately 50% of patients who present to the ED with syncope (although the range is wide **[11,17, 27-37]**) **[Table 1]** are admitted, and this has not been changed with clinical decision rule use. **[38]**

Patients requiring syncope related treatment and some patients with severe coexisting disease or injury caused by the index event may require hospital admission. There is evidence that ED syncope clinical decision/investigation units and/or rapid access syncope clinics are beneficial in achieving the appropriate work-up for high-risk patients **[39,40]** including those with exertional syncope, associated palpitations or suspected device malfunction. **[29]** If an ED syncope clinical decision/investigation unit or a rapid access syncope clinic is not available then high-risk patients are likely to require hospital admission.

### **Clinical decision rules**

There are many ED syncope Clinical Decision Rules (CDRs) and risk-stratification tools that use medical history, examination and ECG findings to stratify patients by their risk of developing both short (i.e. 7-30 day) and long term (i.e. 1 year) serious outcomes. Examples of these are the ROSE rule, San Francisco syncope rule,

OESIL, STePS and the Canadian Syncope Risk Score. [11,15,17,27,32,41] These do not seem to outperform clinical judgment, [42] tend to have low specificity, thus increasing admissions, and have been variably adopted. Some rules and tools have included age. Whilst older patients are undoubtedly at higher risk of adverse outcome after syncope, including age in such tools only reduces their specificity leading to over admission.

There are other guidelines available for use in the ED such as NICE [43]. However the new ESC guidelines are the first to very specifically guide the ED clinician as to which patients should be deemed high risk whilst also attempting to reduce admission rates with alternative investigative strategies (e.g. syncope assessment/decision units and rapid access syncope clinics).

### **Syncope in the elderly**

Syncope is increasingly common with increasing age and is often multifactorial. [44] Although older patients have a wide range of problems likely to cause syncope and do have a higher incidence of underlying cardiac disease, the ED clinician should not refrain from making a diagnosis of reflex or postural syncope in the absence of high-risk features and in the presence of features suggestive of a reflex or postural cause. Although the patient in Case 1 is elderly, there is a prodrome, and a short recovery period and no ischemic or serious conduction disturbances on ECG and reflex syncope is the most likely cause.

### **ED Evaluation**

Specific investigations should only be carried out to answer specific diagnostic questions. An ECG is essential. A completely normal ECG (as opposed to an ECG with non-specific changes) makes a cardiac cause of syncope other than transient arrhythmia less unlikely. 1<sup>st</sup> degree heart block (as seen in case 1) is neither associated with a cardiac or reflex cause of syncope. A bedside or laboratory glucose measurement should be performed to rule out hypoglycaemia, which may present as collapse or seizure.

Measurement of haemoglobin will rule out anaemia (and possible underlying bleeding) as a cause of collapse. Other very selective blood tests may include troponin when cardiac ischaemia-related syncope is suspected and ECG changes are present (see below), and D-dimer when pulmonary embolism is suspected. [2] Serum prolactin has been measured in the past to distinguish between syncope and seizures but is of limited use clinically. No other investigations are routinely required including a chest x-ray and CT brain, which are over ordered in syncope patients.

### **Carotid sinus massage (CSM)**

CSM should be considered in patients over 40 years with reflex syncope of unknown origin (e.g. not situational, related to GTN use, micturition etc) [2]. There is no reason why this cannot be performed in the ED in an area equipped to manage a prolonged pause if the clinician is confident in performing the procedure.

Carotid Sinus Syndrome (CSS) is diagnosed if CSM causes symptomatic bradycardia and/or hypotension in patients with a history and clinical features of reflex syncope. Carotid sinus hypersensitivity is defined by positive CSM without a



syncope history and may be a non-specific finding, being present in 40% of older people. The precise methodology and results of CSM can be found in section 5 of the Practical Instructions for the 2018 ESC Guidelines for the diagnosis and management of syncope. [14]

### **Active standing to measure postural Blood Pressure (BP)**

Classic orthostatic hypotension (time from upright position to abnormal BP response <3 minutes) and delayed orthostatic hypotension (time from upright position to abnormal BP response >3 minutes) can be diagnosed with traditional orthostatic blood pressure measurement. Abnormal BP fall is defined as a progressive and sustained fall in systolic BP from baseline value  $\geq 20$  mmHg or diastolic BP  $\geq 10$  mmHg, or a decrease in systolic BP to  $<90$  mmHg. [2] Other types of orthostatic hypotension exist that are less likely to be detected using standard procedures for orthostatic hypotension: initial orthostatic hypotension (time from upright position to abnormal BP response = 10–15 seconds) and reflex-mediated hypotension (present on prolonged standing).

It is important that active standing is performed by the treating clinician and not delegated to ED nursing staff so that the ED decision maker can carefully observe symptoms and vital signs during the test. Patients who have received fluids may no longer have a positive active stand although, unless extremely symptomatic, TLOC patients should not routinely receive pre-hospital or ED fluid administration. Whilst a negative active stand test in the ED makes orthostatic hypotension less likely as a cause, a patient with a history of persistent syncope with orthostatic features but

normal standard orthostatic BP testing should be referred for specialist opinion so these other types of orthostatic hypotension can be investigated. [14,45].

### **ECG recording**

In addition to the 12-lead ECG, immediate ECG monitoring should be instigated when there is a suspicion of arrhythmic syncope. The new 2018 ESC syncope guidelines [2] support an increased role of prolonged ECG monitoring when arrhythmic syncope is suspected. Establishing a cardiac arrhythmia as the cause of syncope rests on correlating the arrhythmia with symptoms using monitoring devices but these all have significant drawbacks. There is also very little evidence of how long patients suspected of having arrhythmic syncope should be monitored for and various times have been suggested from 24 hours to 28 days.

Cardiac arrhythmia investigation is usually initiated with the Holter monitor but non-compliance and lack of extended monitoring reduces diagnostic yield to less than 20% [2]. Event recorders can monitor over longer periods of time but must be activated and cannot detect asymptomatic arrhythmias. External continuous loop recorders are expensive, require electrodes and bulky recording devices, and produce a large amount of data, which requires sifting. Implantable loop recorders are expensive and necessitate an invasive surgical procedure.

The PATCH-ED study, which used an ambulatory ECG monitor in ED patients with unexplained syncope, identified a symptomatic significant arrhythmia in 1 in 10 patients and a diagnostic finding in 3 in 4. [46] In this study, a third of the significant and symptomatic significant arrhythmias were captured within the first 24 hours

(suggesting a role for prolonged monitoring in the ED or in hospital). The majority of the significant and symptomatic significant arrhythmias were captured in the first 7 days but some significant arrhythmias (mainly non-serious and asymptomatic) were picked up between days 8 and 14.

### **Echocardiography**

Although not routinely required, any patient with a murmur in the context of syncope definitely warrants echocardiography along with any patient with history, physical exam or ECG signs of structural heart disease. This does not need to be done in the ED but could be done in an observation facility or ideally within a few days in an outpatient rapid access syncope clinic. If neither are available then admission for inpatient echocardiography is required. Distinguishing between a benign flow murmur, aortic stenosis and subvalvular obstruction as can be found in HCM, can be difficult. As a rule a shorter (rather than a quieter) ejection systolic murmur is more likely to be benign. The murmur of HCM is unusual in that it becomes louder on standing up (due to decreased venous return reducing the size of the heart). [47]

### **When is a troponin to rule out Acute Coronary Syndrome required?**

Troponin is not required to rule out Acute Coronary Syndrome (ACS) or Myocardial Infarction (MI) unless the ECG shows changes consistent with acute ischaemia [48]. Whilst high sensitivity troponin does have prognostic ability (i.e. it can predict short (1 month) and long term (1 year) risk of serious outcome and death) [49-50] it is not practice changing in clinical practice as yet and should presently only be measured if ACS or MI is suspected.

## **Discharge Instructions and Follow-up**

It is vital that all syncope patients seen in the ED are assessed for and counselled with respect to their fitness to drive, and that this is detailed in their medical notes.

**Figure 5** summarises current United Kingdom (UK) DVLA Fitness to Drive guidelines. Note that guidelines will be very different in every country. In the UK, any patient with suspected cardiovascular syncope, cough syncope or unexplained syncope and any Class 2 (Heavy Goods Vehicle) driver with vasovagal syncope must not drive from the time of their index presentation. These patients should be referred to a syncope specialist to confirm the diagnosis and driving advice.

If low-risk patients require syncope clinic follow-up, there are no guidelines or evidence to suggest the timing of this and these patients should be seen routinely as per local protocols. Again there are no guidelines as to the timing of when high-risk patients requiring syncope clinic follow-up should be seen. If the patient was not seen by a syncope specialist in the ED observation facility or whilst an inpatient this should be on an urgent basis within two weeks.

## **Reflections on case examples**

### *Case 1*

Whilst the ED clinician may be concerned by the patient's age, history of hypertension and 1<sup>st</sup> degree heart block on the ECG, it must be remembered that although older patients have a higher incidence of underlying cardiac disease, reflex or postural syncope is still common. In the absence of high-risk features and in the presence of suggestive features such as the

precipitating lightheaded, diaphoresis and nausea in this case prior to the collapse, a diagnosis of reflex syncope can safely be made.

### *Case 2*

This case highlights two key points. Firstly the absence of suggestive symptoms of reflex or postural syncope and the presence of a high-risk feature (i.e. no pre-warning) this patient must be classed as high-risk syncope. The patient should undergo a period of ED/inpatient monitoring and should be investigated with longer monitoring and echocardiography if there is any history, physical exam or ECG signs of structural heart disease. Secondly and just as important, the patient should be told to refrain from driving at least until the cause of their syncope is explained. Procedures for informing driving authorities is country specific i.e. in the UK the patient has a duty to inform the DVLA whereas in the US the clinician has a duty to report.

### *Case 3*

This case is more challenging. The patient is young, yet suffered syncope without prodrome and significant trauma. An atypical presentation was considered (i.e. there were no signs of underlying cardiac disease yet also a lack of low-risk reflex features including prodrome) and an ambulatory patch monitor was placed. This showed a 26 second pause [**Figure 6**] likely due to non-classical reflex syncope (i.e. reflex syncope without reflex features including prodrome. SA node dysfunction would more likely be associated with an escape rhythm). In view of the severity and regularity of symptoms (2 further episodes had occurred all with

associated trauma subsequent to the index presentation) and the psychological impact of the events, a pacemaker was implanted which halted the episodes.

## **Conclusion**

Syncope is a common ED presentation. The first task is to differentiate syncope from seizure, and, if syncope, rule out an underlying cause. A thorough history of the event and an ECG are essential to determine features suggesting high-risk syncope requiring urgent investigation and admission (or management in a clinical decision or observation unit if available). In cases where benign causes of syncope are suspected, orthostatic BP and carotid sinus massage may be useful. Echocardiography is useful if structural heart disease is suspected; however, troponin is not helpful unless there is a concern for ischemia based on the history or ECG.

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## Table and Figure legends

- Table 1:** Admission rate and composite estimate of short-term (7-30 days) outcomes of patients presenting in ED with T-LOC (Reproduced from Brignole M, et al; 2018 ESC Guidelines for the diagnosis and management of syncope, European Heart Journal 2018; 39 (21): 1883–1948, doi:10.1093/eurheartj/ehy037. **[18]** Reproduced by permission of Oxford University Press on behalf of the European Society of Cardiology.)
- Table 2:** Main categories of causes of syncope grouped by common pathophysiology, presentation and risk.
- Table 3:** ED risk stratification as recommended by the 2018 ESC Guidelines for the diagnosis and management of syncope. **[18]**
- Figure 1:** ED risk stratification flowchart to determine syncope patient management (Reproduced from Brignole M, et al; 2018 ESC Guidelines for the diagnosis and management of syncope, European Heart Journal 2018; 39 (21): 1883–1948, doi:10.1093/eurheartj/ehy037. **[18]** Reproduced by permission of Oxford University Press on behalf of the European Society of Cardiology). SU = syncope unit.
- Figure 2:** ECG showing ARVC. Reproduced from <https://lifeinthefastlane.com/ecg-library/basics/arrhythmogenic-right-ventricular-cardiomyopathy> (accessed 09/08/2018)
- Figure 3:** ECG showing Brugada type 1. Reproduced from <https://lifeinthefastlane.com/ecg-library/brugada-syndrome> (accessed 09/08/2018)

- Figure 4:** ECG showing Classic HCM pattern with asymmetrical septal hypertrophy (reproduced from Kelly BS, Mattu A, Brady WJ. Hypertrophic cardiomyopathy: electrocardiographic manifestations and other important considerations for the emergency physician. Am J Emerg Med. 2007; 25(1): 72-9. Reproduced by permission of Elsevier).
- Figure 5:** Fitness to Drive in TLOC (adapted from A. Hudson, S. Saunder, R. Grant, St. George's University Hospital, London and based on March 2018 UK DVLA advice. 1 = UK Class 1 driver's licence, 2 = UK Class 2 UK Heavy Goods Vehicle driver's licence)
- Figure 6:** Case 3 ECG showing prolonged (26 seconds) pause.

**Table 1**

Author/year/country	Patient s with T-LOC	Number admitted	7-30 day Death	7-30 day non-fatal severe outcome (a)	7-30 day non-fatal severe outcome (a) identified in the ED	7-30 day non-fatal severe outcome (a) identified after initial visit
Costantino, 2008, Italy [27]	676	218 (32%)	5 (0.7%)	36 (5.3%)	n/a	n/a
Brignole, 2006, Italy [28]	465	178 (38%)	6 (1.3%)	n/a	n/a	n/a
Reed, 2010, UK [11]	1100	541 (49%)	17 (1.5%)	79 (7.2%)	n/a	n/a
Ungar, 2015, Italy [29]	295	92 (31%)	1 (0.3%)	n/a	n/a	21 (7.1%)
Birnbaum, 2008, US [30]	713	613 (86%)	4 (0.6%)	57 (8.0%)	32 (4.5%)	25 (3.5%)
Grossman, 2007, US [31]	293	201 (69%)	7 (2.4%)	68 (23%)	56 (19%)	12 (4.1%)
Quinn, 2004, US [32]	684	376 (55%)	5 (0.7%)	79 (11.5%)	n/a	n/a



Quinn , 2006, US [16]	760	448 (59%)	3 (0.4%)	108 (14.2%)	54 (7.1%)	54 (7.1%)
Schladenhaufen, 2008, US [33]	517	312 (60%)	5 (1.0%)	98 (19%)	80 (15.5%)	18 (3.4%)
Sun, 2007, US [34]	477	277 (58%)	n/a	56 (11.7%)	40 (8.6%)	16 (3.4%)
Daccarett, 2011, US [35]	254	118 (46%)	1 (0.4%)	15 (5.9%)	8 (3.1%)	7 (2.8%)
Thiruganasambanda- moorthy, 2014, CAN [36]	505	62 (12%)	5 (1.0%)	49 (9.7%)	22 (4.4%)	27 (5.3%)
Thiruganasambanda- moorthy, 2015, CAN [37]	3662 (b)	474 (13%)	31 (0.9%)	345 (10.3%)	225 (6.7%)	120 (3.6%)
<b>Median</b> <b>(IQR)</b>		<b>49%</b> <b>(32-59)</b>	<b>0.8%</b> <b>(0.6-1.1)</b>	<b>10.3%</b> <b>(7.6-13.0)</b>	<b>6.9%</b> <b>(4.5-10.3)</b>	<b>3.6%</b> <b>(3.4-5.3)</b>

a) Nonfatal severe outcomes generally are defined as a significant new diagnosis, a clinical deterioration, serious injury with recurrence, or a significant therapeutic intervention

b) 3365 patients had 30 day follow-up

**Table 2**

**1. Cardiac syncope (generally high risk)**

- a) Arrhythmia – e.g. bradycardia or tachycardia
- b) Structural – e.g. aortic stenosis, hypertrophic cardiomyopathy, pulmonary embolus

**2. Reflex (neurally-mediated) syncope (generally low risk)**

- a) Vasovagal (VVS)
  - I. orthostatic vasovagal syncope i.e. triggered by standing
  - II. emotional – e.g. triggered by fear or venepuncture
  - III. pain triggered
- b) Situational
  - I. micturition
  - II. gastrointestinal - e.g. swallow syncope, defaecation syncope
  - III. coughing / sneezing
  - IV. post-exercise
  - V. other - e.g. laugh syncope
- c) Carotid sinus syncope
- d) Atypical – i.e. without prodrome / triggers

The above can be predominantly

- Cardioinhibitory reflex syncope - leads to a low cardiac output
- Vasodepressor reflex syncope – leads to a low peripheral resistance
- Mixed – combination of cardioinhibitory and vasodepressor

**3. Orthostatic syncope (generally low risk)**

- a) Drug-induced
- b) Volume depletion
- c) Primary autonomic failure – e.g. Parkinson's disease
- d) Secondary autonomic failure – e.g. diabetes

The above can be exacerbated after exercise, meals or prolonged bed rest due to venous pooling.

OH can be

- Classic (time from upright position to abnormal BP response <3 minutes)
- Delayed (time from upright position to abnormal BP response >3 minutes)

**Table 3**

**Syncopal event**

Low risk:

- Associated prodrome typical of reflex syncope (e.g. light-headedness, feeling of warmth, sweating, nausea, vomiting)
- After sudden unexpected unpleasant sight, sound, smell, or pain
- After prolonged standing or crowded, hot places
- During a meal or postprandial
- Triggered by cough, defaecation, or micturition
- With head rotation or pressure on carotid sinus (e.g. tumour, shaving, tight collars)
- Standing from supine/sitting position

High risk (red flag):

*Major*

- New onset of chest discomfort, breathlessness, abdominal pain, or headache
- Syncope during exertion or when supine
- Sudden onset palpitation immediately followed by syncope

*Minor (high risk only if associated with structural heart disease or abnormal*

*Electrocardiogram; ECG):*

- No warning symptoms or short (<10 s) prodrome
- Family history of Sudden Cardiac Death (SCD) at young age
- Syncope in the sitting position

## **Past medical history**

### Low risk:

- Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode
- Absence of structural heart disease

### High risk (red flag):

#### *Major*

- Severe structural or coronary artery disease (heart failure, low left ventricular ejection fraction; LVEF or previous myocardial infarction)

## **Physical examination**

### Low risk:

- Normal examination

### High risk (red flag):

- Unexplained systolic blood pressure (BP) in the ED <90 mmHg
- Suggestion of gastrointestinal bleed on rectal examination
- Persistent bradycardia (<40 beats per minute; bpm) in awake state and in absence of physical training
- Undiagnosed systolic murmur

## ECG

### Low risk:

- Normal ECG

### High risk (red flag):

#### *Major*

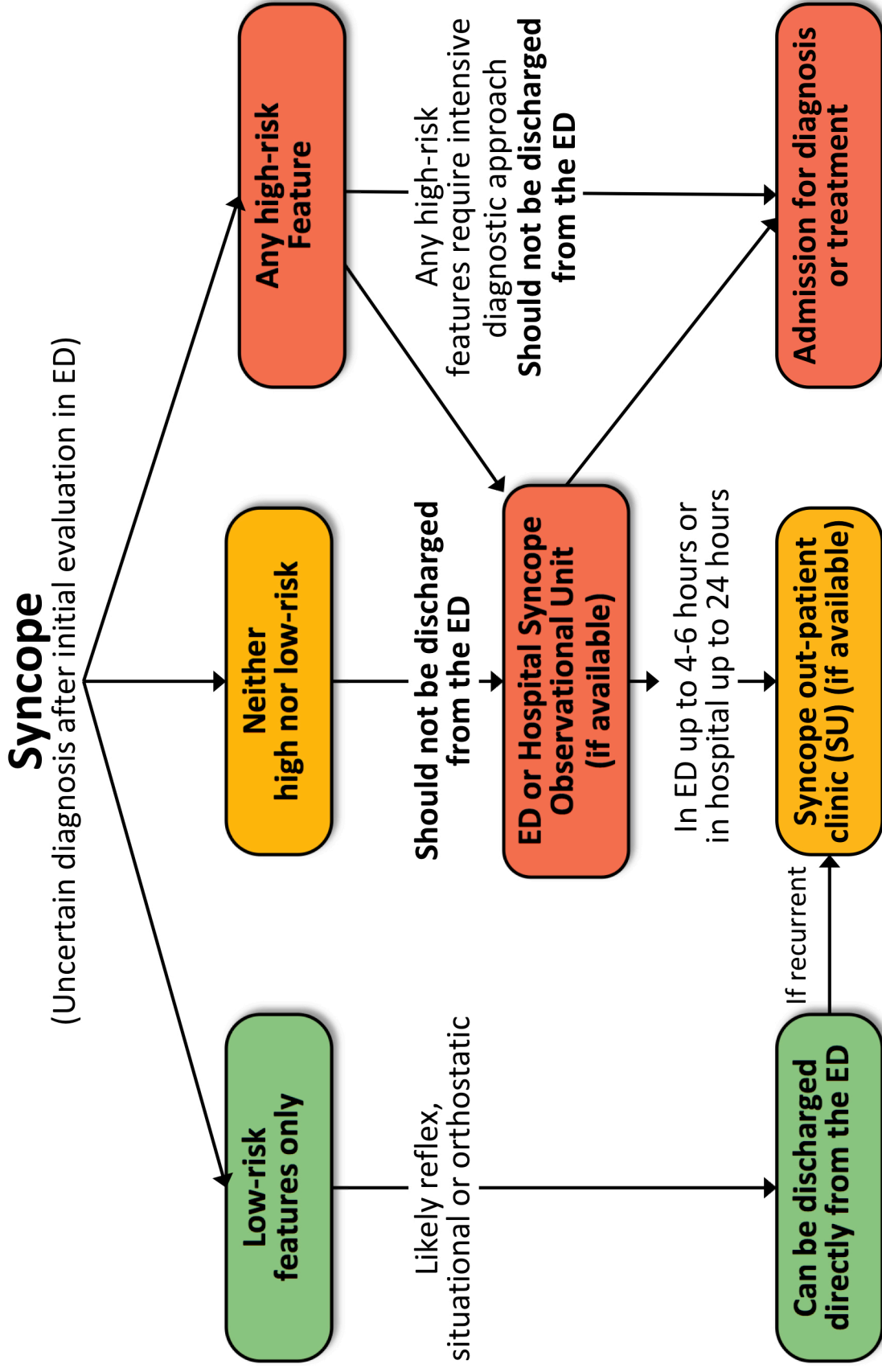
- ECG changes consistent with acute ischaemia
- Mobitz II second- and third-degree atrio-ventricular (AV) block
- Slow Atrial Fibrillation (AF) (<40 bpm)
- Persistent sinus bradycardia (<40 bpm), or repetitive sinoatrial block or sinus pauses >3 seconds in awake state and in absence of physical training
- Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy, or Q waves consistent with ischaemic heart disease or cardiomyopathy
- Sustained and non-sustained Ventricular Tachycardia (VT)
- Dysfunction of an implantable cardiac device (pacemaker or implantable cardioverter defibrillator; ICD)
- ST-segment elevation with type 1 morphology in leads V1– V3 (Brugada pattern)
- QTc >460 ms in repeated 12-lead ECGs indicating long QT syndrome (LQTS)

#### *Minor (high risk only if history consistent with arrhythmic syncope)*

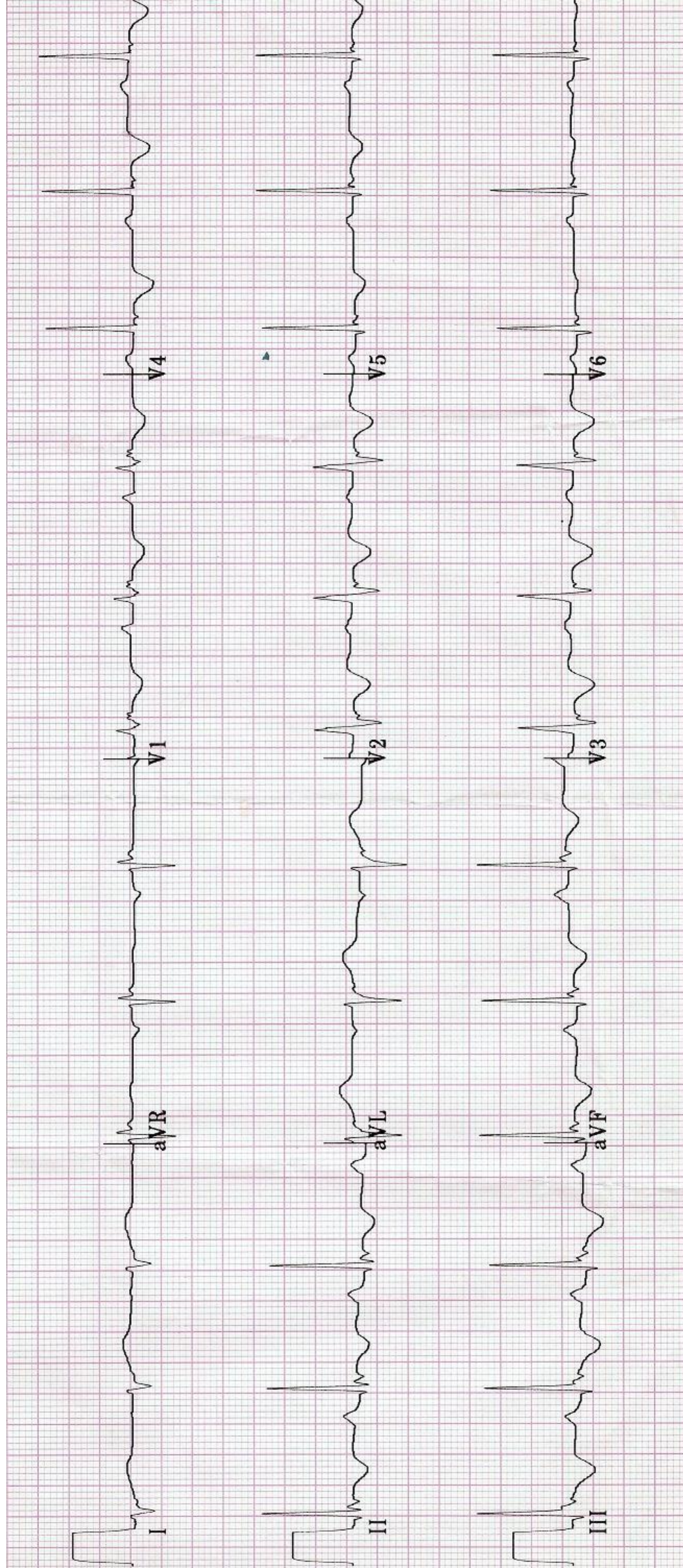
- Mobitz I second-degree AV block and 1° degree AV block with markedly prolonged PR interval
- Asymptomatic inappropriate mild sinus bradycardia (40–50 bpm), or slow AF

(40–50 bpm).

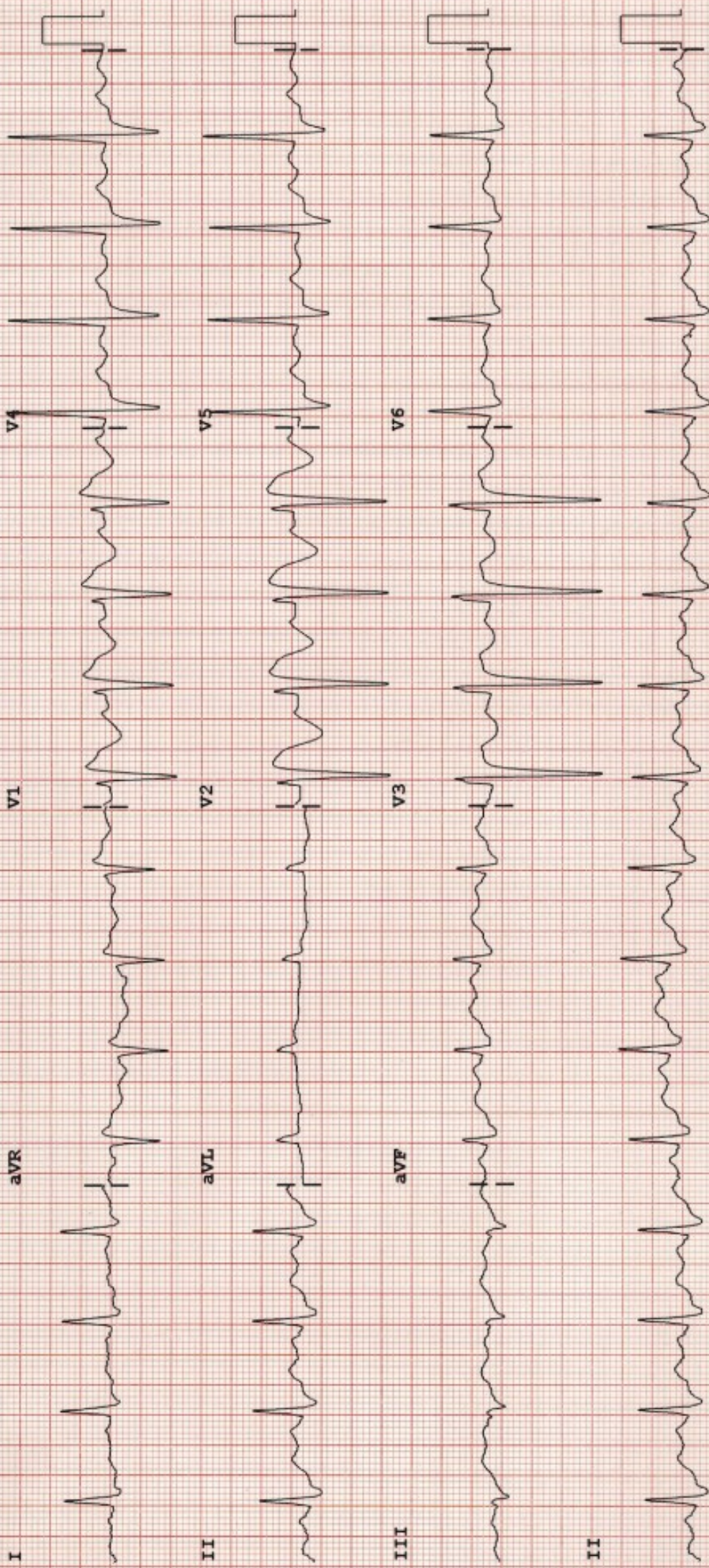
- Paroxysmal supraventricular tachycardia (SVT) or atrial fibrillation
- Pre-excited QRS complex
- Short QTc interval ( $\leq 340$  ms)
- Atypical Brugada patterns
- Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy (ARVC)











Dev: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10 mm/mV F 50~ 0.15-150 Hz PH08 P?





### Key



May drive and need not notify the DVLA



May drive but must notify the DVLA



Must not drive but need not currently inform the DVLA



Must not drive and must inform the DVLA

